Progressive corneal vascularisation as a previously unreported complication of neonatal herpes simplex infection

Christopher J Hammond, Alec F Harden

Bilateral, widespread progressive corneal vascularisation as a result of neonatal herpes simplex infection has not been reported previously. The child presented, one of the first treated with systemic acyclovir, demonstrates the morbidity associated with herpes simplex infection.

Case report

JP was born in January 1982 at 33 weeks' gestation by caesarean section 5 days after spontaneous rupture of membranes with an antepartum haemorrhage. She was initially well, but on day 3 suffered three apnoeic attacks requiring ventilation, and developed bilateral purulent conjunctivitis. On day 5 she developed an erythematous desquamating rash over her scalp which became generalised (Fig 1), mouth vesicles, and pneumonia. Cultures grew herpes simplex type II virus and a diagnosis of disseminated
nated herpes simplex was made. Serial antibodies and maternal status are unavailable. The child was commenced on topical and intravenous acyclovir 6-25 mg three times a day. A cerebrospinal fluid leucocytosis (150x10^3/l) suggested central nervous system involvement. Ocular examination showed a marked bilateral keratitis affecting all corneal levels, initially obscuring iris and other details, shown a few days later in Figure 2.

Her general condition improved and she was discharged at 42 days, with no obvious neurological deficit.

At 1 year examination revealed a small left corneal opacity and visual acuity estimated with a Catford drum was 6/9 in her right eye and 6/12 in the left. She had epiphora, due to an HSV canaliculitis, and repeated vesicular eruptions over all parts of her body, requiring intermittent systemic and topical acyclovir. An intermittent left divergent squint resulted in left amblyopia despite patching, and a Snellen acuity of 6/36, compared with 6/12 on the right.

From 5 years she developed a progressive circumferential bilateral superficial vascularisation with central epithelial scars and opacification ahead of the vessels (Fig 3). This has reduced her vision to 6/60 in the left and 6/36 in the right eye. Typical recurrent HSV keratitis or uveitis have never been observed.

Comment

Neonatal HSV may result in devastating morbidity and mortality; before antiviral therapy 75% of these patients died by 6 months.1 Agents such as acyclovir reduce mortality, but even recent multicentre trials reported 54% mortality in disseminated neonatal HSV infection by 1 year.2

Widespread superficial corneal vascularisation has not been reported after neonatal HSV infection. Acutely, conjunctivitis occurs most frequently (10%), followed by keratitis (7%). Acute series of neonatal HSV suggest 17% ocular involvement,3 but follow up series report up to 60%,4 recognising more retinochoroidal scars, optic atrophy, and squint. Other reported complications include cataracts, phthisis, corneal scarring, recurrent keratitis, and epiphora.

We consider four mechanisms for this clinical picture. First is chronic or recurrent HSV infection. Despite close ophthalmic monitoring, typical HSV keratitis was not seen: no dendrites, faceting, disciforms, or deep vessels appeared and the resulting diffuse superficial vascularisation is atypical.

Second, HSV is a trigger for Stevens-Johnson syndrome as are drugs (unreported for acyclovir). The skin and mucosal lesions, photo-ophthalmia, vessel pattern, and ocular surface disorder are similar to Stevens-Johnson syndrome, as is the progression over years.5 However, keratinisation, metaplastic lashes, and conjunctival scarring were absent.

Third is pemphigoid. Superficial vascularisation and loss of normal corneal epithelium occur, but always accompanied by conjunctival scarring.

Fourth, and perhaps most likely, is loss of limbal stem cells. Surgical or chemical injuries can lead to a progressive superficial vascularisation and loss of normal epithelium if they involve a major portion of the limbus.6 This probably also occurs in Stevens-Johnson syndrome, pemphigoid, and aniridia, but with additional features. Our patient had only corneal features, which suggest a pure syndrome of stem cell loss.
Bilateral angle closure glaucoma and accelerated cataract formation in a patient with AIDS

Naresh Joshi, Peter H Constable, Todd P Margolis, Creig S Hoyt, Timothy J K Leonard

Recent reports have described the occurrence of bilateral angle closure glaucoma secondary to choroidal effusions in patients with AIDS. We report the rapid onset of cataracts in a patient with this syndrome.

Case report
A 44-year-old white man with Pneumocystis carinii pneumonitis, presented with visual deterioration over the course of 1 day. His past ocular history was unremarkable. Visual acuity on presentation was counting fingers at 1 metre in both eyes, which improved to 6/12 with pinhole correction. Anterior segment examination revealed bilateral chemosis and microcystic corneal oedema. The anterior chambers were shallow, especially peripherally; the angles were closed 360 degrees in both eyes on gonioscopy. The pupils were in a mid-dilated position, and reacted to bright illumination segmentally. The intraocular pressure onplanation tonometry was 38 mm Hg (right eye) and 36 mm Hg (left eye). The lenses were clear and the vitreous was quiet. The retinal vasculature was tortuous and dilated, there were scattered cotton wool spots and peripheral retinal haemorrhages, consistent with HIV microvasculopathy. The ora was posteriorly displaced. Bilateral acute angle closure glaucoma was diagnosed.

Treatment was started with intravenous and oral acetazolamide therapy, and bilateral topical pilocarpine 4% and dexamethasone 0.1%. The following day the intraocular pressures remained elevated at 34 mm Hg in both eyes. Bilateral YAG iridotomies were performed, with no appreciable change in anterior chamber depth or change in intraocular pressure. Pilocarpine was discontinued and cyclopentolate 1% therapy begun following the laser procedure. A B ultrasound scan revealed choroidal and ciliary body thickening in both eyes (Fig 1). Within the course of the next 36 hours of instituting cycloplegic therapy the anterior chambers deepened fully and the intraocular pressure decreased to 10 and 12 mm Hg. The following day his intraocular pressures were below 10 mm Hg in both eyes and remained so subsequently. Cortical haze was noted in both lenses: the opacification continued to increase and, within a week, both lenses were completely opaque. The following week the patient underwent cataract extraction and intraocular lens insertion, with a final best corrected visual acuity of 6/9 (right) and 6/6 (left). All the ocular samples removed at the time of surgery including aqueous, cortical aspirate, and the lenticular nuclei from both eyes were examined for cytomegalovirus (CMV) and HIV p24 antigens. These tests were all negative. Serological studies confirmed infection with HIV, with a CD4 count of 32. A significant fourfold rise in CMV antibody titre was detected but no CMV virus was isolated from any source.

Figure 1  B scan ultrasound of left eye demonstrating annular choroidal effusion.